KIDNEY KOLUMNS

Freely filtered from the ISN

KIDNEY KOLUMNS VOL 6 | ISSUE 6 | MAY TO JULY 2026

Dear Readers

Welcome to yet another edition of Kidney Kolumns! This time we bring to you some snippets from the ever expanding world of onconephrology. Although the branch has started gaining popularity in the last decade, it was in 2013 that Dr Abdullah K Salahudden called onconephrology the "latest frontier in the war against kidney disease", leading to widespread use of the term in nephrology circles.

In this edition, we bring to you discussions that are sure to pique the interest of every nephrologist, whether or not onconephrology is a part of their everyday practice.

We also present to you interesting onconephrology experiences from India including the challenges of setting up a dedicated onconephrology clinic in Adyar Cancer institute, kidney transplantation in myeloma and an interesting student contribution on vasculitis in CML.

Lastly, do not forget to tease your neu(ph)rons with our crossword and quiz ! Hope you enjoy reading this edition. As always, we are looking forward to your feedback.

Dr Kartik Ganesh
Dr Pallavi Prasad
(Chief Editors - Kidney Kolumns, June 26)

As always, we welcome your **feedback & contributions** at **education@isn-india.org**

Kidney Kolumns is the official newsletter of the Indian Society of Nephrology (ISN). Launched in 2023, it is published quarterly and serves as a vibrant platform for the nephrology community to exchange knowledge, share updates, and foster professional growth.

The newsletter currently accepts contributions by invitation only. All content is reviewed by the editorial board prior to publication. However, student contributors are encouraged to submit their work without an invitation and should contact the student editors for further guidance.

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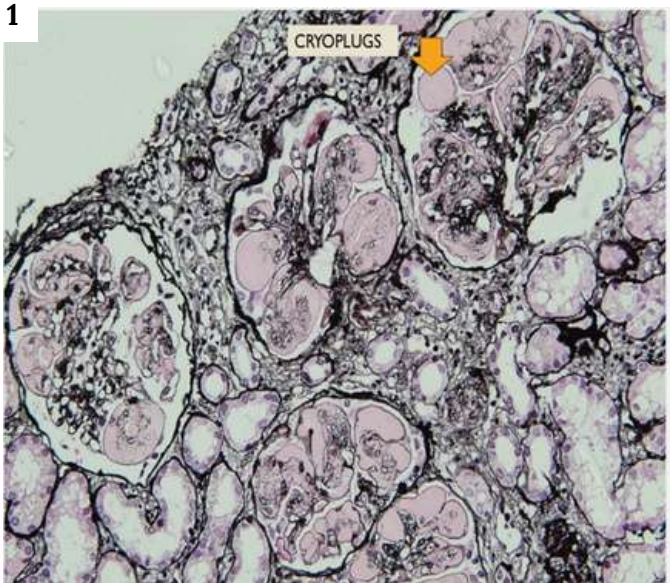

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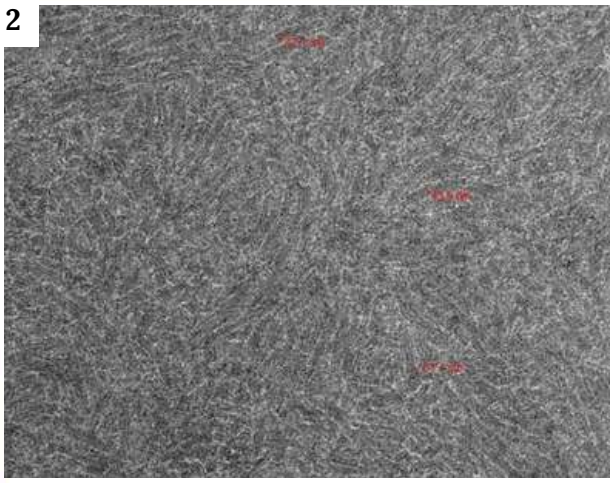

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Dr Pallavi Prasad
Associate Professor, Department of Nephrology, Vardhaman Mahavir Medical College & Safdarjung Hospital, New Delhi

GUEST EDITOR

Dr Kartik Ganesh
Department of Nephrology & Kidney Transplantation, VPS Lakeshore Hospital, Kochi

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DM, FISN, FISOT, FASN, FRCP (London)
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Director & Head, Department of Nephrology, Medanta - Medicity, Gurgaon

DESIGN

Dr Aniruddha Datta
DNB, DM, Senior Resident
Nephrology, IPGMER, Kolkata

PUBLISHER

Indian Society of Nephrology
Department of Nephrology & Renal Transplant, Medanta - The Medicity
Choudhary Baktawar Singh Road, Sector 36, Gurugram 122001
Ph No: +91 9810383522
E-mail: education@isn-india.org

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Renal Pathology

IMAGE 1
Kidney biopsy light microscopy at 40X—sections stained by silver methenamine showing silver-negative cryoplugs

IMAGE 2
EM kidney biopsy - 10,000X - curved microtubules in cryoglobulin deposits

Cover Image Credits

Dr Geetika Singh
Professor, Division of Nephropathology, Department of Pathology, AIIMS, New Delhi

Kidney Kolumns

SECRETARY'S DESK

Dear Members

Greetings from the ISN Secretariat!

My congratulations to our young team of Kidney Kolumns for bringing out the society's quarterly newsletter – **The Kidney Kolumns**. The Theme of this issue is `**OncoNephrology**,' which is a rapidly expanding subspeciality of nephrology, and I congratulate the guest editor, Dr Kartik Ganesh, for bringing the content beautifully.

Dr Shyam B Bansal
Honorary Secretary, Indian SN

I would also like to update our members about some of the society's recent activities and ISNCON 2026 Kochi -

1) ISNCON 2026 KOCHI:

The 55th Annual Conference of the Indian Society of Nephrology, ISNCON 2026, is going to take place between 17th to 20th December 2026 at Kochi Convention Centre, Hotel Grand Hyatt, Kochi Balgotty, Kerala. Kochi, often referred to as the "Queen of the Arabian Sea," is a vibrant coastal city known for its natural beauty, rich cultural heritage, and warm hospitality. You can explore the venue by clicking at www.isnconkochi.com. I want to highlight some of the key attractions of ISNCON 2026.

- **ISN Workshops (17th December) -** like preceding years, the Indian SN would be doing multiple workshops for the benefit of our students and delegates. We have brought some new webinars this year featuring renowned National and International faculty. You can register for one of the workshops.
- **Intervention Nephrology**
- **Critical Care Nephrology and POCUS**
- **Finance, Medicolegal and AI**
- **Histopathology and Immunogenetics**

Registration for Workshops is Complimentary; however, seats are limited, and each person can register for only one workshop.

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Abstract Submission for ISNCON: I request all members to upload their abstracts on the website at www.isn-india.org. The last date of abstract submission is 31st July.

There would be many renowned national and international faculty members, as in previous years, to maintain the quality of the scientific program.
Registration for ISNCON 2026 has started. You can register by visiting the ISN website and going to the registration page: www.ISN-India.org.

2) WEBINAR WITH ISGD

The Indian SN of Nephrology recently conducted a **webinar on Advances in Podocytopathy in collaboration with ISGD**. On 29th May, the webinar recording is available on the Indian Society of Nephrology's website.

3) MEMBERSHIP OF THE INDIAN SOCIETY OF NEPHROLOGY

I would request everyone to become members of the Indian SN to avail the benefits of membership

- The membership is lifetime, with only one-time payment of Rs 5000 for nephrologists and only 4000 for students. Students can become members after the first year of completion of their DM/DNB. You can go to the website, fill out the form, and make the payment online to become a member (QR code below)
- Members can access the Indian Journal of Nephrology
- Discounted registration fee in ISNCON
- Membership is essential to present their abstracts in ISNCON.
- Nowadays, after our collaboration with ERA, all members of the Indian Society of Nephrology can also become ERA members by paying only Rs 500/- and avail all benefits of ERA, including access to journals and discounted registration for the ERA meeting.
- Membership is essential if young nephrologists want to be part of various teams of Indian SN - Young nephrology team for Kidney Kolumns, Social media team and others.

I would request that everyone encourage their students/colleagues who are not ISN India members to become members.

Looking forward to seeing you in the beautiful City of Kochi in December.

KIDNEY TRANSPLANTATION IN ACTIVE MULTIPLE MYELOMA

Dr Kartik Ganesh, *Consultant, Nephrology and Renal Transplantation*
Dr Abi Abraham M, *HOD, Nephrology and Renal Transplantation*
Dr Govind Gangadharan, *Associate Consultant, Department of Oncology*
VPS Lakeshore Hospital and Research Center, Kochi

The Editorial.

Multiple myeloma (MM) has historically been a deterrent to renal transplantation primarily due to poor survival of both the patient and the allograft. Kidney involvement with multiple myeloma is common and includes cast nephropathy, monoclonal immunoglobulin deposition disease, amyloidosis and fibrillary and immunotactoid glomerulopathy. MM patients developing ESRD have poor survival, with studies reporting a mean survival of 18.3 months. Hence, kidney transplant strategies in myeloma are understandably complex and fraught with multiple questions- low overall survival being the most important factor. However there has been a demonstrable improvement in long term survival of MM with the introduction of newer chemotherapeutic agents and regimens, which can be applied in transplanting patients of MM with ESRD. All experiences are limited to small case reports. One strategy is to attain either complete or very good partial remission following induction therapy using novel agents, followed by high-dose autologous stem cell transplant (ASCT) and then a kidney transplant. Another proposed strategy is combined ASCT and kidney transplant from the same donor , which offers the advantage of not requiring significant post-transplant immunosuppression owing to utilization of both grafts from the same donor. However, this approach is logistically complex and

Dr Kartik Ganesh, *Department of Nephrology and Kidney Transplantation, VPS Lakeshore Hospital, Kochi, Kerala*

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not widely available given the requirement for an HLA-matched donor for both organs.

Given the complex nature of these strategies, a kidney transplant without ASCT is an attractive option. We discuss here two cases of Kidney transplantation in multiple myeloma from our centre.

CASE 1

A 50-year-old male with autosomal dominant polycystic kidney disease and end stage renal failure on dialysis was evaluated for a kidney transplant. On routine evaluation, he was found to have a monoclonal band in the Beta 1 microglobulin region with kappa free light chain restriction and 30% plasmacytosis. His skeletal survey was unremarkable. He was initiated on a bortezomib based regimen while continuing dialysis. A repeat Bone Marrow after 6 months showed reduction in plasmacytosis (17%), kappa light chain levels were in normal limits and M band was undetectable. A living donor kidney transplantation was performed. Pre transplant CDC cross match was negative and anti HLA antibody titres were acceptable. Induction used was Basiliximab and maintenance immunosuppression included tacrolimus, steroids and fortnightly bortezomib. He was kept off mycophenolate and steroids were tapered to minimal dose within 6 weeks. He was followed up on this regimen for 2 years with no incidents of rejections, BK virus or cytomegalovirus (CMV) infections. S. Creatinine at 2 years was 1.4mg/dl.

CASE 2

A 56-year-old male underwent a kidney transplant 25 years ago and was re-initiated on dialysis once the graft failed due to chronic antibody mediated rejection. Workup for a re-transplantation revealed MM with a positive M band (1g/dL), kappa restriction and 15% plasmacytosis on bone marrow examination. His skeletal survey was unremarkable. He was initiated on a bortezomib based regimen while continuing dialysis. A repeat Bone Marrow biopsy after 6 months showed reduction in plasmacytosis (9%), kappa light chain levels were within normal limits and M band was undetectable. He underwent a living donor kidney transplantation. Pre-transplant CDC cross match was negative and the anti HLA antibody titers were acceptable. Induction used was Basiliximab and maintenance immunosuppression included tacrolimus, low dose mycophenolate , steroids and fortnightly bortezomib. Steroids were tapered to minimal dose within 6 weeks. He was followed up on this regimen for 2 years with no incidents of rejections, BK virus or cytomegalovirus (CMV) infections. S. Creatinine at 2 years was 1.2mg/dl.

In general kidney transplants in MM have inferior graft survival. In a fairly large published cohort of MM patients undergoing kidney transplantation, graft survival at 1 and 5 years was 82.5% and 66%, respectively. Progression-free survival rates of the cohort at 1, 3, and 5 years were 83.3%, 55.6%, and 44.4%, respectively. Risk stratification of MM patients who will do well in the long term remains imperfect in identifying individuals with a greater likelihood of long-term survivorship and graft survival. High-risk cytogenetic abnormalities are the most widely recognized prognostic indicators of poor outcomes in MM. The detection by FISH of t (4;14), t (14;16), del(17p), 1q gain [gain(1q)], and 1p deletion [del(1p)] has been consistently associated with both lower

disease-free survival and overall survival despite intensive therapy.

In both the cases described above, could be attained in 6 months from the start of bortezomib based chemotherapy and they were cleared for transplantation by the oncologists. Non T cell depleting induction (Basiliximab) was used for both patients and maintenance immunosuppression was modified as well. Case 1 was maintained off mycophenolate, whereas Case 2 was kept on a minimal dose of mycophenolate keeping in mind his sensitised status. Both were kept on maintenance bortezomib twice a week. 2 year graft survival with this regimen was good with no disease recurrence in the graft. There were no documented rejections or significant infections (including CMV and BK virus). These transplantations are not without precedent with few published cases achieving significant graft survival.

Kidney transplantation in patients with multiple myeloma require careful selection of patients and documentation of CR or VGPR before attempting transplant. These are defined as follows:

COMPLETE RESPONSE (CR)
negative immunofixation on both serum and urine, disappearance of any soft tissue plasmacytomas, and <5% plasma cells in the bone marrow aspirate.

VERY GOOD PARTIAL RESPONSE (VGPR)
Residual M-protein detectable only by immunofixation but not electrophoresis, or a ≥90% reduction in serum M-protein with urine M-protein < 100 mg/24 h.

MEASURABLE RESIDUAL DISEASE
(earlier called minimal residual disease or MRD) **NEGATIVITY**
Absence of clonal plasma cells on next-generation flow cytometry or sequencing with sensitivity ≥ 10^{-5}. It is now considered the most sensitive marker of long-term prognosis and represents the optimal hematological status for kidney transplant eligibility.

The optimal timing for undergoing kidney transplantation in MM is yet to be defined. Prolonged waiting periods reduce the number of quality-of-life years, whereas proceeding too early might cause relapse and graft dysfunction. A waiting period of 6–12 months after HSCT and remission is often recommended, depending on risk stratification- 6 months for standard risk MRD negative patients and 12 months for Standard-risk, MRD-positive patients or high-risk, MRD-negative patients. High-risk, MRD-positive patients without remission are unlikely to benefit from kidney transplantation.

Significant challenges remain. Although 2 year survival has been uneventful in our 2 patients, duration of bortezomib therapy is not indefinite. Graft survival after stoppage of bortezomib and the response of the dyscrasia remains to be seen.

Although still not recommended, there may exist a template to transplant selected patients of MM in partial remission on bortezomib without a preceding ASCT and achieve reasonable short-term survival. Long term results are still unknown.

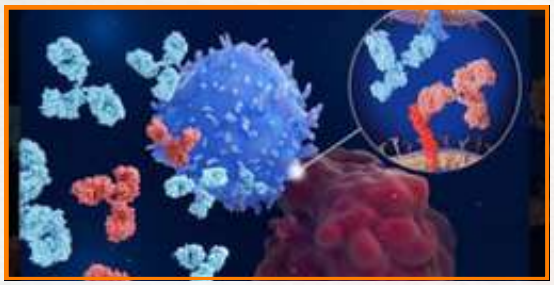
KK Quiz

OncoNephrology Challenge

Dr Vineet Behera

Q 1 Which cancer treatment introduced in 2011 revolutionized oncology but unexpectedly created a new subspecialty of nephrology "Onconephrology"?

A) Rituximab
B) Checkpoint inhibitors
C) Bortezomib
D) Imatinib



Q 2 Which journal first coined the term "Onconephrology" as a distinct discipline?

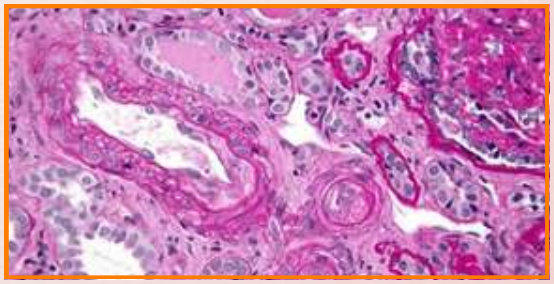
A) JASN
B) Kidney International
C) CJASN
D) NEJM

Q 3 Which chemotherapy drug was nicknamed the "magnesium thief" because of profound renal magnesium wasting?

A) Vincristine
B) Cisplatin
C) Cyclophosphamide
D) Gemcitabine

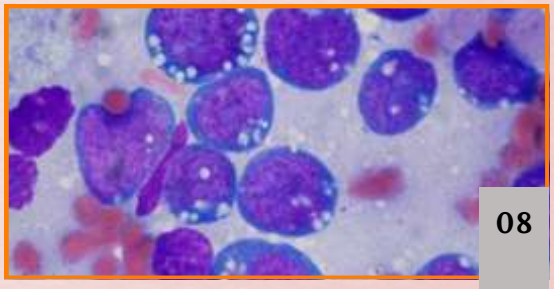
Q 4 Which chemotherapeutic drug is classically associated with thrombotic microangiopathy?

A) Cisplatin
B) Gemcitabine
C) Cyclophosphamide
D) Vincristine



Q 5 Which syndrome was first described in children with Burkitt lymphoma after chemotherapy?

A) Fanconi syndrome
B) Tumor lysis syndrome
C) TTP
D) SIADH



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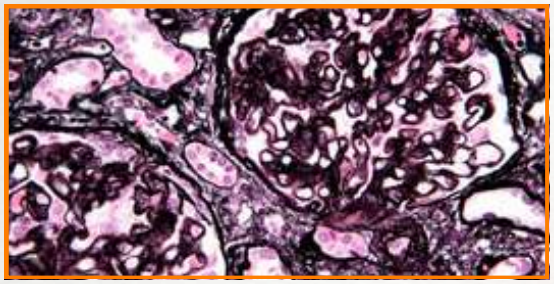
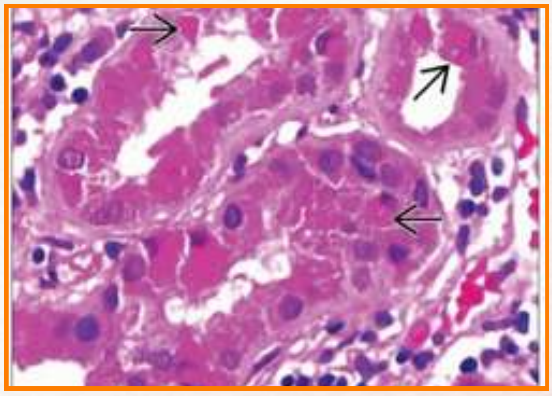
Q 6 Which malignancy is most commonly associated with membranous nephropathy?

A) Renal cell carcinoma **C)** Colon carcinoma
B) Lung carcinoma **D)** Prostate carcinoma



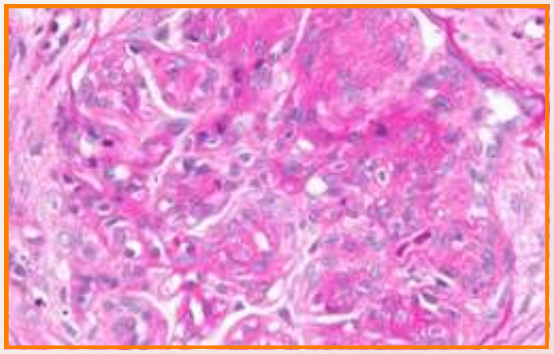
Q 7 Which chemotherapeutic agent commonly causes Fanconi syndrome?

A) Ifosfamide **C)** Vincristine
B) Cyclophosphamide **D)** Paclitaxel



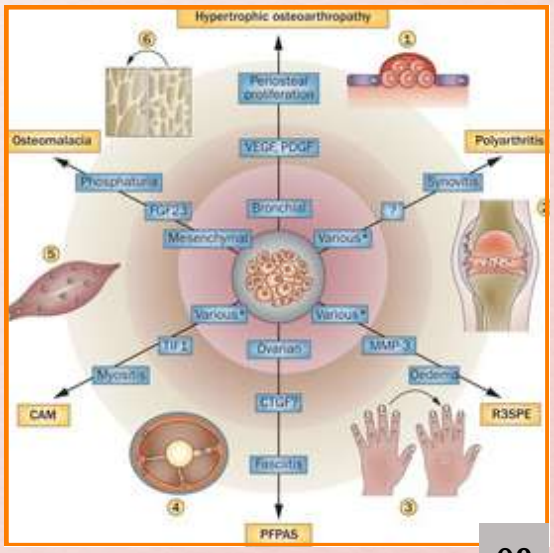
Q 8 Which hematologic malignancy is classically associated with membranoproliferative GN due to cryoglobulinemia?

A) Multiple myeloma **C)** Acute leukemia
B) Chronic lymphocytic leukemia **D)** Hodgkin lymphoma



Q 9 Which cancer is known as the "internist's tumor" because of numerous paraneoplastic syndromes?

A) Lung cancer **C)** Colon cancer
B) Renal cell carcinoma **D)** Pancreatic cancer



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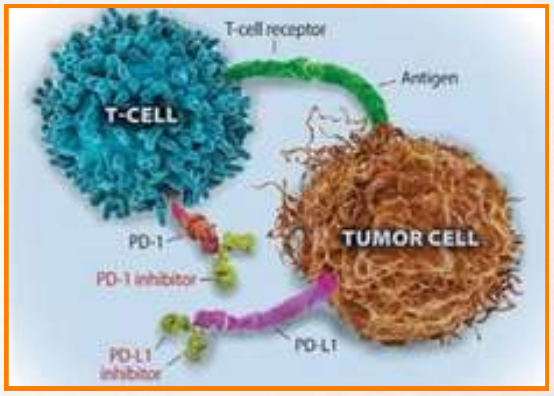
Q 10 Which of the following cancers most commonly causes hypercalcemia mediated by PTHrP?

A) Squamous cell carcinoma of lung
B) Prostate cancer
C) Thyroid carcinoma
D) Chronic leukemia

Q 11 Which immune checkpoint inhibitor is most frequently associated with acute interstitial nephritis?

A) Nivolumab
B) Rituximab
C) Daratumumab
D) Lenalidomide

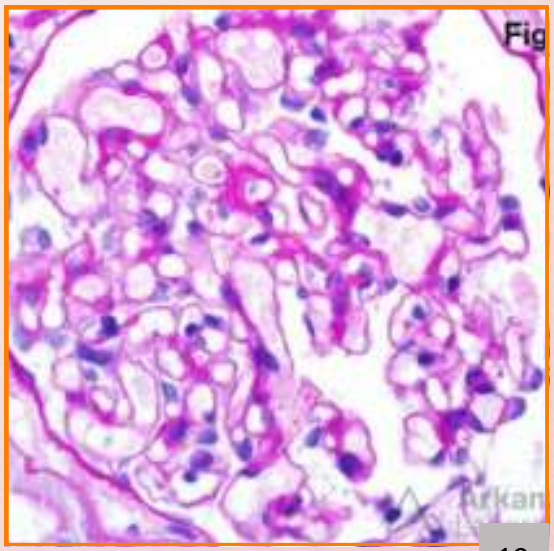


Q 12 Which drug used for multiple myeloma can itself cause thrombotic microangiopathy?

A) Daratumumab
B) Bortezomib
C) Carfilzomib
D) Cyclophosphamide

Q 13 Which malignancy is most strongly associated with paraneoplastic minimal change disease?

A) Hodgkin lymphoma
B) Colon carcinoma
C) Lung carcinoma
D) Melanoma



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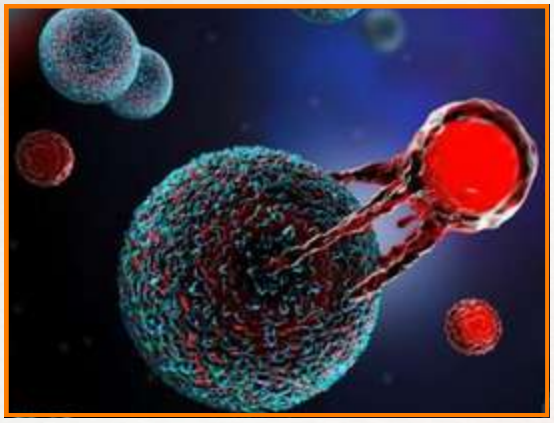
Q 14 Which type of renal injury is most commonly observed on biopsy in CAR-T-associated AKI?

A) Crescentic GN
B) Minimal change disease
C) Acute tubular injury
D) Membranous nephropathy



Q 15 Which malignancy is called the "great imitator" in nephrology because it can produce almost every renal lesion?

A) Renal cell carcinoma
B) Multiple myeloma
C) CLL
D) Lymphoma

KK Quiz
Answers @ Pg 34

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Immune Checkpoint Inhibitor-Associated

ACUTE KIDNEY INJURY: STRATEGIES FOR THERAPEUTIC RECHALLENGE

Dr Mayuri Trivedi, *Senior Consultant and Head, Department of Nephrology, Lokmanya Tilak Municipal General Hospital, Mumbai*
Dr Naman Jain, *DrNB (Nephrology) SR, Department of Nephrology, Lokmanya Tilak Municipal General Hospital, Mumbai*

Immune checkpoint inhibitors (ICIs) have fundamentally altered the therapeutic landscape of modern oncology, significantly improving survival outcomes across a diverse spectrum of malignancies. However, the clinical utility of ICIs is frequently constrained by immune-related adverse events (irAEs) secondary to a systemic loss of peripheral tolerance against autoantigens, which triggers autoimmune damage across multiple organ systems (Xu et al, 2024). While nephrotoxicity is less frequent than other barrier-surface or endocrine irAEs —such as enterocolitis, thyroiditis, and cutaneous toxicities [Fujii et al, 2017- acute kidney injury (AKI) remains a significant clinical concern. Overall AKI incidence ranges from 9.9% to 29% in patients receiving immunotherapy, with true ICI-associated AKI (ICI-AKI) accounting for approximately 2.2% to 7.1% of these cases. (Zhou et al, 2024 ; Manohar et al, 2019).

ICI-AKI typically presents as a low-grade injury, and its incidence varies according to the specific immunotherapeutic regimen utilized; notably, the lowest incidence is observed with programmed death-ligand 1 inhibitors anti-PD-L1) (Zhou et al, 2024). Histopathologically, the predominant diagnostic finding on renal biopsy is acute interstitial nephritis (AIN) or acute tubulointerstitial nephritis (ATIN). Key risk factors predictive of recurrent nephrotoxicity include a shorter latency period to the initial AKI event, concurrent exposure to concomitant nephrotoxic agents (e.g., proton pump inhibitors [PPIs], non-steroidal anti-inflammatory drugs [NSAIDs], and antibiotics), and specific histopathological features like AIN or

ATIN. (Zhou et al,2024)

Although acute episodes of ICI-AKI are managed effectively using first-line corticosteroid therapy or second-line immunosuppression, the optimal strategy regarding the resumption of immunotherapy following renal recovery remains controversial. In a multi-center cohort study by Gupta et al., 28.2% of 429 patients with ICI-AKI underwent an immunotherapeutic rechallenge, resulting in a 16.5% recurrence rate of ICI-AKI (Gupta et al, 2021). Recurrent injury typically manifested at a median of 10 weeks post-rechallenge. Notably, patients selected for rechallenge initially presented with milder stages of AKI during their primary episode. While 180-day survival rates did not differ significantly between patients who were rechallenged and those who permanently discontinued the drug, the cohort that developed recurrent ICI-AKI post-rechallenge exhibited significantly higher mortality.

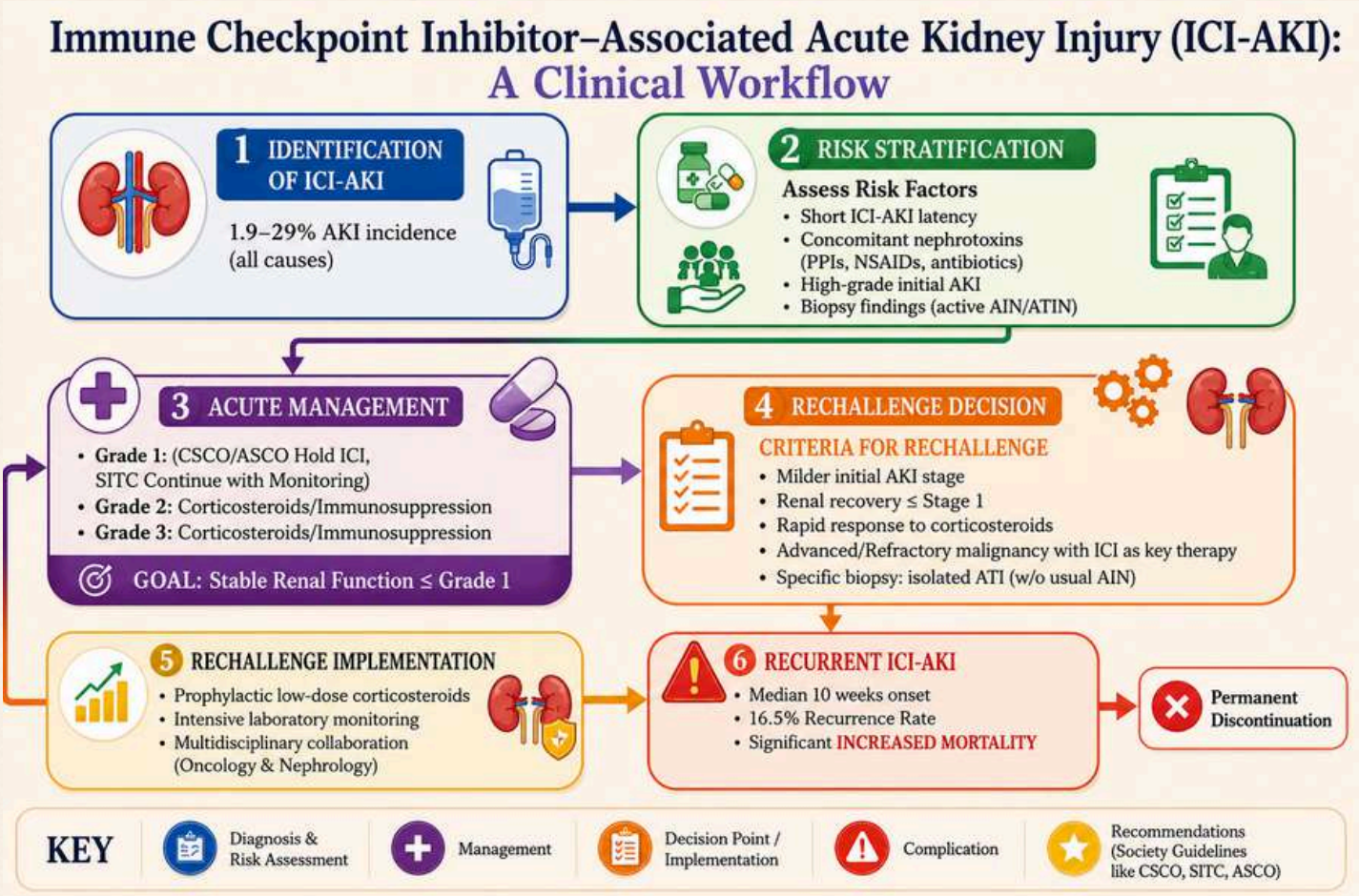
Emerging evidence suggests that specific patient subsets may be safely considered for ICI resumption once kidney function recovers to stage 1 AKI. Favourable candidates include patients whose renal biopsies reveal isolated acute tubular injury (ATI) without conventional ICI-associated lesions, as well as those with AIN who demonstrated a robust, prompt therapeutic response to baseline corticosteroid treatment (Zhou et al, 2024). Additionally, the administration of low-dose prophylactic corticosteroids during the rechallenge phase represents a viable strategy to mitigate the risk of recurrent nephrotoxicity. (Manohar et al, 2020).

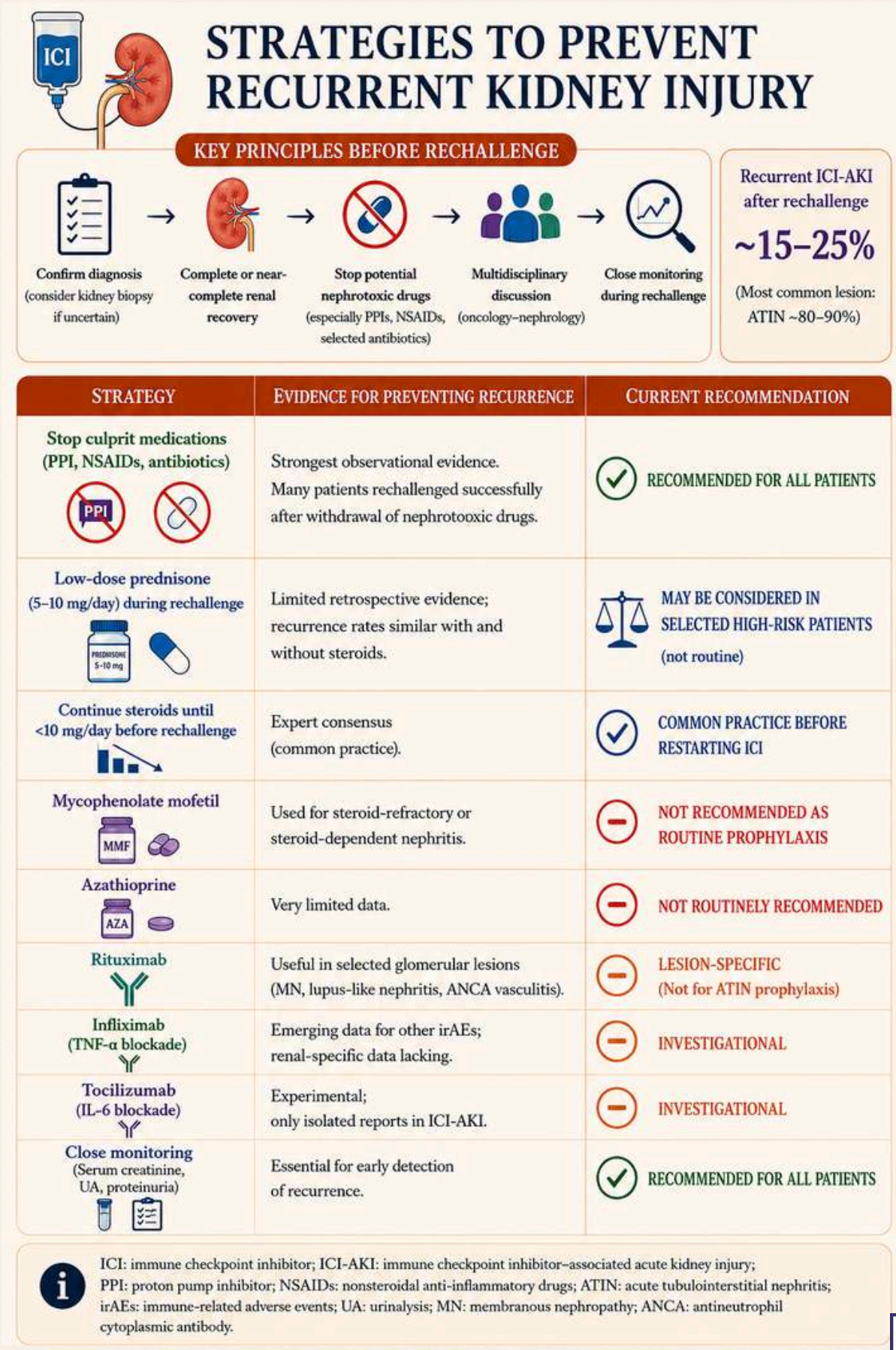
International society guidelines currently demonstrate substantial heterogeneity regarding recommendations for ICI rechallenge following a renal irAE. For Common Terminology Criteria for Adverse Events (CTCAE) Grade 1 nephrotoxicity, the Society for Immunotherapy of Cancer (SITC) guidelines support the continuation of therapy under rigorous clinical surveillance. Conversely, the Chinese Society of Clinical Oncology (CSCO) and the American Society of Clinical Oncology (ASCO) advocate for temporary drug suspension, permitting a rechallenge only after serum creatinine returns to baseline values.

For CTCAE Grade 2 events, consensus exists across all major guidelines to consider an ICI rechallenge once the toxicity de-escalates to less than CTCAE Grade 1 alongside stable renal function. For CTCAE Grade 3 nephrotoxicity, the CSCO, SITC, and National Comprehensive Cancer Network (NCCN) guidelines recommend an individualized, case-by-case evaluation. This approach balances the anticipated oncological benefit against the risks after a mandatory 2-month washout period, provided all renal symptoms have completely resolved. In contrast, ASCO guidelines mandate the permanent discontinuation of ICIs if a CTCAE Grade 3 event is directly attributed to the immunotherapeutic agent. For CTCAE Grade 4 toxicities, all societies uniformly recommend permanent treatment discontinuation. (Tang et al, 2025).

In summary, definitive consensus data regarding the safety of ICI rechallenge following ICI-AKI are lacking, necessitating highly individualized clinical decision-making. Resumption of therapy is primarily justified in patients with advanced or refractory malignancies where ICIs represent

the definitive or solitary therapeutic option, and where complete renal recovery to less than Grade 1 has been documented. Ultimately, a prudent clinical approach demands formal multidisciplinary collaboration between oncology and nephrology teams, intensive laboratory monitoring, and the strategic deployment of low-dose corticosteroid prophylaxis. While permanent treatment discontinuation is more common in severe, biopsy-confirmed cases of AIN, it should not be applied as an absolute mandate.




ICI
STRATEGIES TO PREVENT RECURRENT KIDNEY INJURY
KEY PRINCIPLES BEFORE RECHALLENGE
Confirm diagnosis (consider kidney biopsy if uncertain)
Complete or near-complete renal recovery
Stop potential nephrotoxic drugs (especially PPIs, NSAIDs, selected antibiotics)
Multidisciplinary discussion (oncology–nephrology)
Close monitoring during rechallenge
Recurrent ICI-AKI after rechallenge
~15–25%
(Most common lesion: ATIN ~80–90%)
STRATEGY
EVIDENCE FOR PREVENTING RECURRENCE
CURRENT RECOMMENDATION
Stop culprit medications (PPI, NSAIDs, antibiotics)
PPI
Strongest observational evidence. Many patients rechallenged successfully after withdrawal of nephrotoxic drugs.
RECOMMENDED FOR ALL PATIENTS
Low-dose prednisone (5–10 mg/day) during rechallenge
PREDNISONE 5-10 mg
Limited retrospective evidence; recurrence rates similar with and without steroids.
MAY BE CONSIDERED IN SELECTED HIGH-RISK PATIENTS (not routine)
Continue steroids until <10 mg/day before rechallenge
Expert consensus (common practice).
COMMON PRACTICE BEFORE RESTARTING ICI
Mycophenolate mofetil
MMF
Used for steroid-refractory or steroid-dependent nephritis.
NOT RECOMMENDED AS ROUTINE PROPHYLAXIS
Azathioprine
AZA
Very limited data.
NOT ROUTINELY RECOMMENDED
Rituximab
Useful in selected glomerular lesions (MN, lupus-like nephritis, ANCA vasculitis).
LESION-SPECIFIC (Not for ATIN prophylaxis)
Infliximab (TNF-α blockade)
Emerging data for other irAEs; renal-specific data lacking.
INVESTIGATIONAL
Tocilizumab (IL-6 blockade)
Experimental; only isolated reports in ICI-AKI.
INVESTIGATIONAL
Close monitoring (Serum creatinine, UA, proteinuria)
Essential for early detection of recurrence.
RECOMMENDED FOR ALL PATIENTS
ICI: immune checkpoint inhibitor; ICI-AKI: immune checkpoint inhibitor–associated acute kidney injury; PPI: proton pump inhibitor; NSAIDs: nonsteroidal anti-inflammatory drugs; ATIN: acute tubulointerstitial nephritis; irAEs: immune-related adverse events; UA: urinalysis; MN: membranous nephropathy; ANCA: antineutrophil cytoplasmic antibody.

Establishing An OncoNephrology Programme

IN A DEVELOPING COUNTRY: FROM CONCEPT TO CLINIC

Dr Rimda Wanchoo, *Professor of Medicine Director of Quality, Division of Kidney Diseases and Hypertension Medical Director, Julia and Israel Waldbaum-Davita Dialysis unit, USA*
Dr Rajeevalochana Parthasarathy, *Senior Consultant Nephrology, Medway Hospitals, Chennai*

With the advent of novel cancer therapeutics, patients with cancer are living longer. However, improved survival has been accompanied by an increasing burden of kidneyrelated complications, including acute kidney injury (AKI), chronic kidney disease (CKD), electrolyte disorders, proteinuria, hypertension, treatment related nephrotoxicity and dialysis planning. The development of dedicated onconephrology consultations in North America and Europe has demonstrated the benefits of multidisciplinary care, early nephrology involvement, rapid treatment, and systematic data collection. While these models provide valuable lessons, implementation in low and middle income countries including India requires adaptation to local realities, including workforce limitations, delayed referrals, financial constraints and uneven access to specialized services (Table 1).

In such settings, the goal should be to build a layered and scalable model of care. A successful onconephrology program should begin with integration rather than infrastructure. Early growth is often driven more by building relationships with oncology teams than by creating standalone nephrology services. Working closely with oncology and integrating nephrology into existing tumor boards, chemotherapy review meetings, and inpatient workflows creates early visibility. Referral pathways should be intention

-ally simple, initially focusing on a few high-yield indications such as AKI, CKD, electrolyte disorders, proteinuria and uncontrolled hypertension. Structured referral criteria and rapid access consultation slots encourage oncologists to involve nephrology earlier in the disease course rather than as a rescue service. Equally important is maintaining responsiveness, as timely kidney interventions frequently determine whether cancer therapy can continue uninterrupted. These relatively simple steps can generate substantial impact without requiring major capital investment.

Sustainability requires transitioning from isolated consultations to an integrated system of care. Starting with a periodic clinic (for example weekly) and gradually increasing frequency based on demand is often more practical than launching a resource-intensive model from the outset. Systematic tracking of referrals, kidney outcomes, treatment modifications, dialysis requirements, and follow-up rates helps demonstrate value to hospital leadership while creating opportunities for quality improvement and research. Continuous education of oncologists, nurses, residents, pharmacists, intensivists, and trainees remains central to building awareness and strengthening collaboration and ensuring long-term program success.

Inresource-limited environments, task sharing can facilitate earlier recognition of kidney complications and timely nephrology referral, helping prevent interruptions in cancer therapy. Telenephrology and virtual multidisciplinary clinics can extend expertise to remote cancer centres and satellite units, while simple referral checklists and electronic templates improve efficiency and continuity of care. Where access to kidney biopsy, dialysis, or advanced diagnostic services is limited, partnerships with tertiary nephrology centers may offer a more feasible and cost-effective alternative to developing these capabilities locally.

International partnerships can substantially accelerate implementation. Programs such as the International Society of Nephrology-Sister Renal Centers (ISN–SRC) model demonstrate how mentorship, case-based learning, protocol sharing, faculty exchange, and collaborative research can strengthen local expertise while remaining responsive to regional needs and resource constraints. Importantly, measures of success in developing countries should extend beyond patient volume and include indicators such as reduction in treatment interruptions, earlier nephrology referral, improved follow-up retention, prevention of severe AKI, and preservation of access to cancer therapies.

The following experience from a tertiary cancer center in India illustrates how these principles can be adapted in a resource-constrained setting:

At Adyar Cancer Institute, Chennai, we established a dedicated onconephrology service in 2022 in response to the growing need for integrated kidney care in patients with cancer. Early challenges included limited awareness among oncologists, delayed referrals , workforce constraints, and demonstrating value in a resource-limited setting. Through dedicated clinic sessions and closer integration with oncology services, referrals gradually increased and more patients were followed longitudinally for kidney related complications. Standardized referral pathways were subsequently

introduced for common indications such as AKI, CKD, proteinuria, electrolyte disorders, treatment-related nephrotoxicity, and dialysis planning. Regular meetings and educational sessions were conducted with oncologists, residents, and nursing staff to establish clear referral criteria for nephrology consultation, facilitating timely recognition of kidney-related complications. Standardized protocols were developed for the management of dialysis dependent patients, including the introduction and implementation of both peritoneal dialysis (PD) and continuous kidney replacement therapy (CRRT) for appropriate clinical scenarios. A kidney biopsy service was established within the cancer center, enabling timely tissue diagnosis, with all specimens reviewed by an experienced nephropathologist to ensure accurate clinicopathological correlation. The onconephrology team also became an active participant in multidisciplinary tumor boards and mortality and morbidity meetings, providing the nephrology perspective on complex cases while simultaneously gaining a deeper understanding of oncologic decision making, treatment priorities, and patient outcomes. Regular onconephrology continuing medical education (CME) sessions were conducted to enhance awareness and strengthen collaboration between nephrologists and oncologists. These sessions were centered around real-world case discussions, multidisciplinary deliberations, and expert opinions that helped guide complex treatment decisions while promoting evidence-based kidney care in patients with cancer. To facilitate timely communication, a dedicated WhatsApp group was established involving nephrologists, oncologists, and trainees. This platform enabled rapid discussion of acute kidney-related issues, streamlined referrals, facilitated follow-up of shared patients, and allowed prompt exchange of investigation results and management plans.

An important milestone was the establishment of the ISN–SRC collaboration with Northwell Health's onconephrology specialists in 2023. This partnership helped strengthen clinical and academic capacity and coincided with substantial growth in referrals and follow-up care. Since then, follow up rates have nearly doubled from approximately 20% to 44%, reflecting a transition from episodic consultation to longitudinal kidney care integrated within cancer management.

Our experience suggests that successful onconephrology programs are driven less by infrastructure and more by strong interdisciplinary partnerships, continuous education, and early integration of nephrology into cancer care.

TABLE I: Key Components for Establishing and Scaling an OncoNephrology Programme in Resource-Limited Settings / New Programmes

Components	Start-up Phase	Growth Phase	Practical Tip for Developing Countries
Institutional Support	Administrative approval, oncology alignment	Dedicated Onconephrology unit	Start as a shared oncology-nephrology service

Components	Start-up Phase	Growth Phase	Practical Tip for Developing Countries
Leadership	Nephrology and oncology champions	Multidisciplinary leadership team	Identify local champions early
Referral System	Simple referral criteria and forms	Standardized pathways and EMR	Use practical referral triggers
Clinic & Workforce	Weekly clinic, nephrologist, nurse support	Dedicated clinic days and coordinators	Start with half-day/week and cross-train staff
Clinical Services	Manage AKI, CKD, hypertension, proteinuria and therapy related side effects	Specialized clinics (GN, transplant, survivorship)	Focus on common consult indications
Diagnostics & Dialysis	Basic labs, imaging, dialysis access	Biopsy, oncology dialysis, PD/CRRT	Build referral networks before expansion
Protocols & MDT	Local SOPs and regular case discussions	Disease-specific pathways and tumor board integration and participate in MDT's	Adapt guidelines to available resources
Training & Telemedicine	Staff education and virtual consults	Structured training and outreach	Use short teaching sessions
Data & Research	Registry and outcome tracking	Prospective databases and collaborative studies	Collect core data from day one
Sustainability	Hospital support and partnerships	Grants and program expansion	Demonstrate clinical value and scale gradually

Magnesium Matters

REDUCING CISPLATIN-ASSOCIATED ACUTE KIDNEY INJURY IN ROUTINE ONCOLOGY CARE

A COMMENTARY *on Gupta S et al. JAMA 2025.IV Magnesium and Cisplatin Associated AKI*

Dr Veenaa Manjari S, *Consultant, Department of Nephrology, Medway Hospitals, Chennai*

What if one of the simplest and inexpensive interventions could reduce one of the most common toxicities of a life-saving chemotherapy drug? Cisplatin remains indispensable in the treatment of many cancers, but its use is frequently complicated by acute kidney injury. Although hydration has traditionally been the cornerstone of preventing cisplatin-induced kidney injury, magnesium supplementation is emerging as a simple and promising addition. Gupta et al, in a landmark multicentre retrospective cohort study, explored the association between prophylactic intravenous magnesium administration and the risk of cisplatin-associated AKI (CP-AKI). Their results have renewed interest in the role of magnesium as a nephroprotective agent.

Why Magnesium? The Biological Rationale for Kidney Protection

Preclinical studies suggest that magnesium protects against cisplatin nephrotoxicity through multiple mechanisms. Magnesium deficiency reduces expression of drug efflux transporters on the apical surface of proximal tubular cells, leading to increased accumulation of platinum within these cells. It increases production of inflammatory cytokines and reactive oxygen species which leads to inflammatory injury and oxidative stress within the kidney. Magnesium supplementation reverses these effects by promoting platinum excretion and reducing tubular injury.

Most previous studies on CP-AKI have been single centre studies with small sample sizes,hence limiting the strength of evidence in favour of prophylactic magnesium.

Study Design and Population

The study was a multicentre retrospective cohort analysis conducted at five major U.S. cancer centres, including Brigham and Women's Hospital, Memorial Sloan

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Kettering, and MD Anderson Cancer Center. It included 13,719 adult cancer patients treated with their first dose of intravenous cisplatin between 2006 and 2022. The median age was 59 years, and 57% were male. Of these, 3,893 patients (28.4%) received prophylactic IV magnesium on the first day of cisplatin chemotherapy, while 9,826 did not. The median magnesium dose was 2 grams.

Primary Outcome and Results

The primary composite outcome was CP-AKI defined as a 2-fold or more increase in serum creatinine from baseline or initiation of kidney replacement therapy or death within 14 days of cisplatin initiation.

CP-AKI or death occurred in 104 of 3,893 patients (2.7%) receiving IV magnesium. In contrast, 520 of 9,826 patients (5.3%) without magnesium experienced this outcome. The adjusted odds ratio was 0.80 (95% CI, 0.66–0.97), indicating a 20% reduction in risk.

Secondary Outcomes and Sensitivity Analyses

Results remained consistent across multiple sensitivity analyses and secondary outcomes, including alternative definitions of CP-AKI or death and major adverse kidney events at 90 days. The investigators used inverse probability treatment weighting and multivariable adjustment to account for differences between patients who received magnesium and those who did not, including demographics, baseline investigations, cisplatin dose, and concurrent nephrotoxic therapies.

Insights from Subgroup Analysis

Benefit of magnesium was most pronounced in young patients, females, those with preserved kidney function (eGFR ≥90 mL/min/1.73 m^2) and those with higher baseline serum magnesium levels.

Clinical Implications

This study provides strong clinical evidence that prophylactic intravenous magnesium may reduce the risk of cisplatin-associated acute kidney injury. Magnesium is inexpensive, widely available, and safe. Patients with preserved kidney function and higher baseline magnesium levels appeared to derive the greatest benefit, suggesting that magnesium may be most effective when administered prophylactically before significant magnesium depletion or kidney injury occurs, rather than as a treatment after toxicity has already developed.

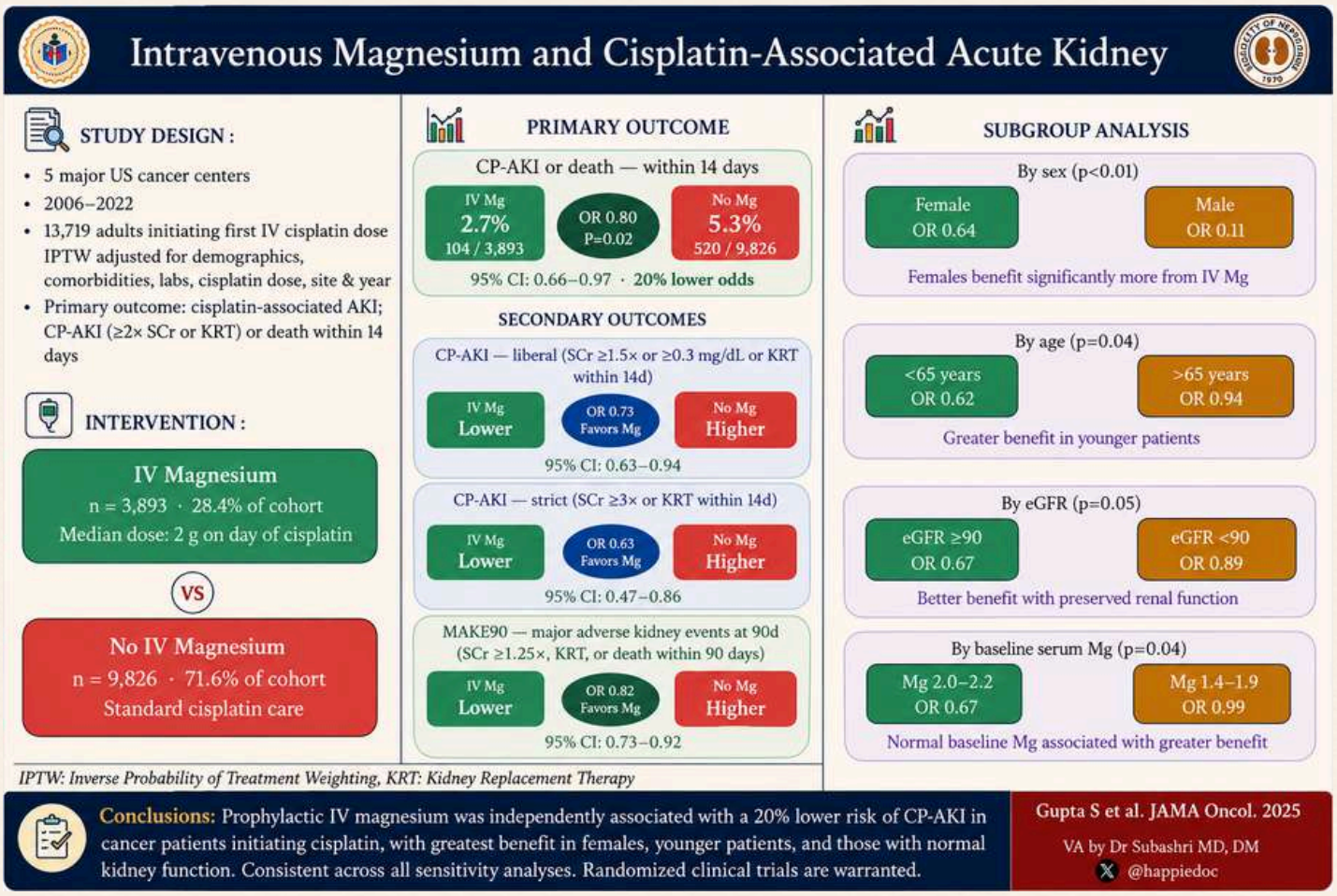
Limitations

Authors have acknowledged limitations inherent to observational studies. Residual confounding cannot be completely excluded, and treatment allocation was not randomized. Hence, causality cannot be definitively established. Additionally, the study does not address optimal magnesium dosing, timing or duration of prophylaxis.

Conclusion

Gupta et al have highlighted the potential role of magnesium not just as a replacement for electrolyte loss, but as a possible shield against cisplatin nephrotoxicity. While further randomized studies are needed, this study suggests that a small, inexpensive measure could have a meaningful impact on reducing cisplatin-related kidney injury and improving patient care.

Magnesium Matters: Reducing Cisplatin-Associated Acute Kidney Injury in Routine Oncology Care



Anticancer Drug Dosing in Kidney Dysfunction

WHAT EVERY NEPHROLOGIST SHOULD KNOW ABOUT IT

Dr Syeda Hurmath, *Consultant Nephrologist, Trustwell Hospital, Bangalore*

As nephrologists, we are increasingly involved in the care of patients with cancer. Whether managing acute kidney injury (AKI), electrolyte abnormalities, chronic kidney disease (CKD) dialysis support, or onconephrology consultations, our role continues to expand. One recurring challenge is determining how anticancer drugs should be prescribed in patients with impaired kidney function. Historically, dosing recommendations have varied across package inserts, drug handbooks, and institutional protocols, often leading to inconsistent clinical decisions. The recently published Anticancer Drug Dosing in Kidney Dysfunction (ADDIKD) guideline aims to address this gap by providing a standardized, evidence-based approach to anticancer drug dosing in kidney disease.

ADDIKD is more than a drug-dosing guideline, it represents a shift in philosophy. Traditionally in renal disease, oncology practice often focused on a single question: "What is the creatinine clearance (CrCl)?" ADDIKD encourages clinicians to ask a broader question: "What is the most accurate assessment of kidney function, and how can we safely deliver the most effective cancer treatment?" The guideline recognizes that preventing toxicity is important, but not at the cost of compromising potentially curative therapy. Treatment intent, patient performance status, concomitant nephrotoxic medications, and the reliability of kidney function assessment must all be considered before altering a chemotherapy dose.

Moving Beyond Cockcroft-Gault

Perhaps the most practice-changing

aspect of ADDIKD is its recommendation to move away from routine reliance on the Cockcroft-Gault (CG) formula. Developed nearly five decades ago, the CG equation has several limitations. It was derived from a small cohort, predominantly male, uses total body weight, and performs poorly in elderly patients, obese individuals, amputees, patients with sarcopenia, cachexia, paraplegia, or extremes of body composition. In oncology patients, where muscle wasting is common, CG may substantially overestimate or underestimate true kidney function, leading to inappropriate dose reductions or unnecessary avoidance of effective therapies. ADDIKD therefore aligns oncology practice with modern nephrology by recommending estimated glomerular filtration rate (eGFR) calculated using the CKD-EPI equation and classification according to Kidney Disease: Improving Global Outcomes (KDIGO) CKD stages. The guideline also revives the importance of directly measured GFR (mGFR). In selected situations including high-dose methotrexate, cisplatin, carboplatin, and patients in whom eGFR is unreliable, mGFR remains the preferred method of assessment.

Drug	Historic Guidance Pitfall	ADDIKD Strategic Approach
Methotrexate	Empirical 50% dose cuts below 50 ml/min across all settings.	Mandates mGFR for high doses. Maintains full doses for curative intent at eGFR 45-59 ml/min.
Cisplatin	Broad, rigid contraindications when CrCl < 40-50 ml/min.	Aligns with KDIGO: permits split-dosing strategies to preserve treatment efficacy.
Carboplatin	Heterogeneous kidney function inputs into the Cockcroft-Gault CrCl.	Prefers mGFR, mandates BSA-adjusted eGFR over CrCl when using estimation models.
Nivolumab	Lack of guidance regarding emerging sub-clinical nephrotoxicity data.	Recommends baseline urinalysis/electrolytes and strict monitoring for immune-related AKI.

Individualize Dose Reduction

ADDIKD challenges the traditional practice of automatic chemotherapy dose reduction solely based on reduced kidney function. Instead, it advocates an individualized approach that considers treatment intent, patient performance status, availability of alternative therapies, concomitant nephrotoxic medications, and the potential impact of dose modification on cancer outcomes. Consequently, two patients with identical eGFR values may require very different treatment strategies; a fit patient receiving potentially curative therapy may benefit from maintaining optimal drug exposure, whereas

a frail patient receiving palliative treatment may warrant a more conservative approach. This patient-centered framework is one of the key strengths of the guideline.

Lessons from Key Drugs

The ADDIKD recommendations for methotrexate, cisplatin, carboplatin, and nivolumab reinforce a common theme (Table1), anticancer drug dosing in CKD should be individualized rather than driven by arbitrary kidney function thresholds. Across these agents, the guideline emphasizes accurate assessment of kidney function, consideration of treatment intent, and balancing toxicity against treatment efficacy. Importantly, some drugs may require measured GFR for optimal dosing, while others may not require dose adjustment despite impaired kidney function but still warrant close nephrology surveillance because of their potential nephrotoxicity.

The Take-Home Message

ADDKID reinforces several principles familiar to our specialty:

- Accurate kidney function assessment matters.
- eGFR should be interpreted thoughtfully.
- Measured GFR remains valuable in selected patients.
- Treatment decisions should be individualised.
- Multidisciplinary collaboration improves outcomes.

The ADDIKD guideline also has a quick reference tool useful for both oncologists and nephrologists for anticancer drug dosing. Most importantly, the guideline recognises that kidney function assessment is not merely a laboratory exercise. It directly influences cancer treatment, toxicity, and survival.

KK Crossword

NEPHRONS & NEOPLASIA

KK Crossword

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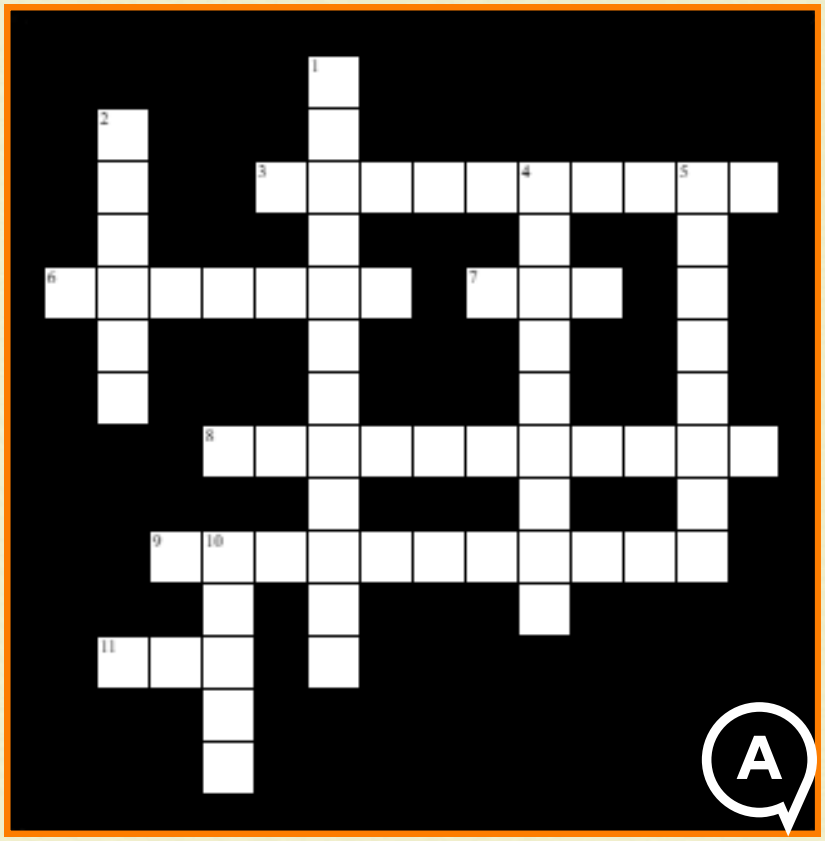
MAY TO JULY 2026



Solve the crossword ONLINE

KK Crossword Answers @ Pg 34

1 Electrolyte wasting commonly seen with Cetuximab

2 This formula is used to calculate the optimum dose of Carboplatin

3 This precision-engineered cell therapy, which enriches for regulatory T-cells, aims to mitigate GVHD following allogeneic HCT

4 This anticancer drug isolated from Catharanthus roseus (Madagascar periwinkle) can cause SIADH

5 The phase III KEYNOTE 426 study was the first to compare the efficacy of pembrolizumab and this TKI for advanced clear cell RCC

6 The most common renal related adverse effect of VSPIs (VEGF Signalling Pathway Inhibitors)

7 Which of the solid organ tumors is most commonly associated with AA amyloidosis?

8 German pathologist and bacteriologist historically credited with publishing the first accurate clinical description of 'radiation nephropathy"

9 Monoclonal antibody used to treat severe cytokine release syndrome associated with T-cell engaging bispecific antibodies

10 This type of myeloma is associated with an increased serum anion gap

11 The first checkpoint inhibitor approved by the FDA

12 The earliest and most frequent target organ in Graft-versus-host disease

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5 FASTEST FINISHERS OF NEPHRONS & NEOPLASIA Crossword

Congratulations to our brilliant Speedy Solvers!!!

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5	Muthu Subramaniyan Shankar	@Dr_Sms_11

Thank you to everyone who participated and made Nephrons & Neoplasia Crossword a grand success!

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Renal Limited Vasculitis

IN A PATIENT WITH CONCURRENT HEMATOLOGICAL MALIGNANCY

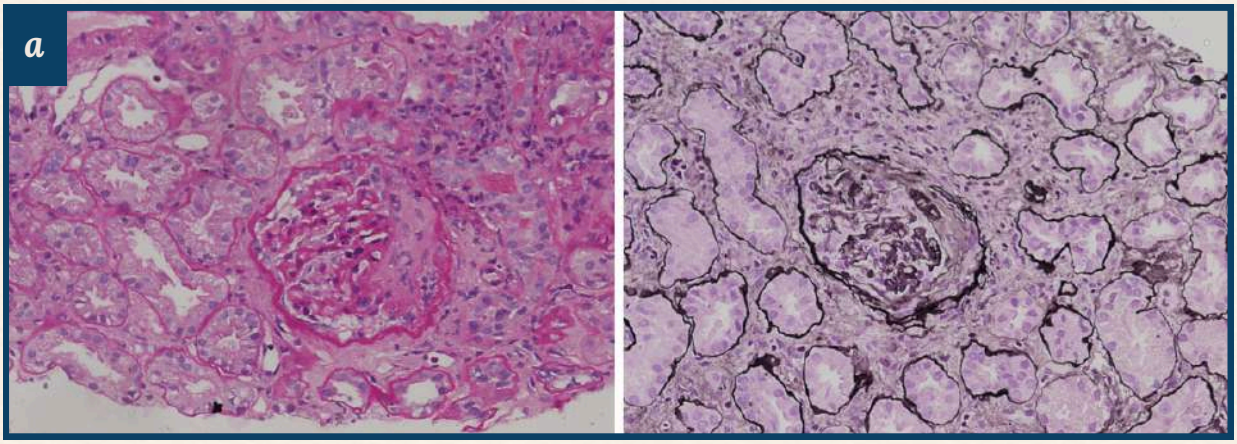
Dr Raghav Gupta, Dr Dipankar Bhowmik
Department of Nephrology, AIIMS New Delhi

A 55-year-old gentleman with no pre-existing co-morbidities, presented with complaints of dyspnoea and decreased urine output of 7 days duration. He had leucocytosis (TLC 14,120/µL) and deranged kidney function (urea 439 mg/dL, creatinine 10.2 mg/dL), requiring haemodialysis for fluid overload and advanced azotaemia. Ultrasonography showed normal-sized kidneys with preserved corticomedullary differentiation. Urinalysis revealed 2+ proteinuria with 100–120 RBCs/HPF. Serological workup including ANA, ANCA (IIF), SPEP, free light chains, complement levels (C3, C4, total complement), hepatitis B/C, HIV, blood and urine cultures were unremarkable. Echocardiography excluded endocarditis. CRP was mildly elevated (37 mg/L) with near-normal procalcitonin. Kidney biopsy revealed pauci-immune focal necrotizing glomerulonephritis with fibrocellular crescents(Figure 1a). After excluding active infection, pulse methylprednisolone was administered followed by IV cyclophosphamide (15 mg/kg) and oral steroids. Kidney function recovered remarkably, achieving dialysis independence within one week. Persistent neutrophilic leucocytosis prompted haematological evaluation. PET-CT demonstrated generalised FDG-avid lymphadenopathy on both sides of the diaphragm. Peripheral blood RT-PCR detected BCR-ABL transcripts (82%), and bone marrow biopsy(Figure 1b)confirmed chronic phase of chronic myeloid leukaemia (hypercellular marrow, marked myeloid proliferation, dwarf megakaryocytes, no excess blasts). Imatinib was initiated but switched to dasatinib due to gastrointestinal intolerance. He received two monthly IV cyclophosphamide pulses with steroid taper and was subsequently lost to follow-up but continued dasatinib. At last follow-up, BCR-ABL transcripts had declined to 33%, white cell counts normalised, and serum creatinine was 1.46 mg/dL. We decided to observe the renal functions in subsequent visits and no maintenance therapy for renal vasculitis was started.

To our knowledge this is the first ever reported case of renal-limited ANCA negative pauci-immune glomerulonephritis diagnosed concurrently with CML. Glomerular diseases are well recognised in haematological

malignancies — MCD in Hodgkin lymphoma, MPGN in CLL — and vasculitis has been reported in both haematolymphoid and solid organ malignancies, although it more commonly involves skin, CNS, or peripheral nerves rather than the kidney (Zhang et al, 2025; Salvarani et al, 2018; Sonne et al, 2020; Loricera et al, 2013). Systemic vasculitis in this setting is rare, generally ANCA-positive, and renal-limited vasculitis is rarer still. A bidirectional association between ANCA-associated vasculitis and haematological malignancies has been proposed (Folci et al, 2019); however, our patient was distinctly ANCA-negative with isolated renal involvement. The mechanisms of the vasculitis associated with malignancy are complex, including formation of immune complexes, polyclonal activation of B lymphocytes, antibodies directed against endothelial antigens, and direct vascular infiltration by neoplastic cells (Mertz et al, 1992; Nashitz et al, 1995). The diagnosis of both illnesses was made concurrently and CML can be postulated to be a driving factor in the causation of renal vasculitis. Infection-related glomerulonephritis was excluded by normal complement levels, absence of clinical infection, and lack of immune deposits on biopsy. The sustained renal recovery following treatment of both vasculitis and CML supports a paraneoplastic aetiology.

This case provides important insights into the important but rare association of vasculitis in hematological malignancies and the importance of kidney biopsy and early initiation of therapy directed towards vasculitis and the underlying malignancy.



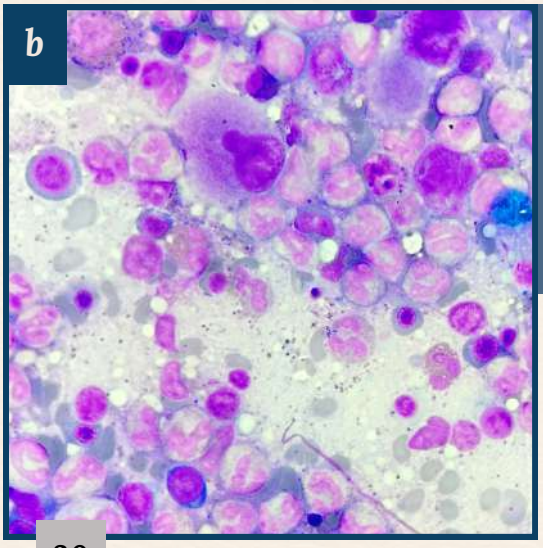


Figure 1.a. Kidney Biopsy (LM:PAS, Silver methanamine) Showing Fibrocellular Crescents

Figure 1.b. Bone Marrow Showing Myeloid Predominance and Dwarf Megakaryocyte

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ECO-Kidney: A New Arrow

IN THE QUIVER OF ABO INCOMPATIBLE KIDNEY TRANSPLANTATION

Dr Sai Spandana Gollapudi
Senior Resident, Department of Nephrology, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Puducherry

It was a match not meant to be
A barrier in scarlet between O, A and B

A battle against antibodies, fierce and deep
Fought by columns, filters and drugs flung in heaps

Within the success of transplant, magnificent and glorious
A creeping shadow looms, maleficent and vicious

The villain of pneumonia advances each day
Tearing down the lungs all along its way

As the little lass fights a losing battle
Dreary echoes of sepsis rattle

Until the rise of the ECO-kidney that chimed
Every hurdle shall be bravely climbed

EChOing to mankind a gift of science
That indeed resculpted a perfect alliance!

Kidney transplantation is the treatment of choice for patients with end-stage renal disease from both a clinical and economic standpoint. Yet, ABO incompatibility remains a glaring barrier to expansion of donor pool, especially in developing nations.

In the last two decades, ABO incompatible kidney transplant (ABOiKT) has emerged as a highly promising strategy to mitigate the gap and substantially increase the living donor pool. This is especially true for individuals of blood group O or B, who typically have longer waiting periods in terms of access to compatible grafts. Although ABOiKT has been shown to provide excellent graft outcomes, the procedure itself is complicated by stringent and elaborate pre-conditioning protocols involving potent immunosuppressive agents, anti-CD20 monoclonal antibody, antibody removing techniques such as plasmapheresis or immunoadsorption to attain a target pre-transplant antibody titer. This often has far-reaching consequences, in the form of higher risk of infections, hemorrhage and malignancy in the post-transplant setting. Very often, it compounds the psychological burden associated with a renal transplant, in

terms of prolonged hospital stay, emergency re-admissions and financial instability.

The inspiration for this poem stems from the tragic loss of a young female patient at a tertiary center in South India, who underwent an ABO incompatible kidney transplantation with her father as the donor, but later succumbed to severe pneumonia resulting from extensive immunosuppression.

Recent research by from China and from UK has explored techniques using enzymes to modify ABO blood group antigens in the kidney, thus converting A and B organs to the universally compatible O group (Enzyme Converted kidney). The core concept that the only apparent difference between A, B and O RBCs resides in the presence or absence of either GalNAc or Gal on the terminal H-antigen raised the possibility that A and B RBCs could be converted to O RBCs if that sugar could be selectively removed. Such enzyme-converted O RBC organs might then be usable as universal donors, rising above the ABO barrier.

This strategy is aimed at expanding the arena of ABOiKT by mitigating the risks involved in the routine use of potent pre-conditioning regimens. A further consideration is to help allow full use of the growing deceased donor organ supply to match the continuing demand for compatible organs. It could also potentially pave the way for direct clinical translation into reduced waiting times and equity of organ distribution for patients with end-stage kidney disease. Few studies in the last couple of years have demonstrated that enzymatic conversion of both A and B group kidneys to O group (ECO-kidney) using machine perfusion techniquescan mitigate hyperacute antibody-mediated injuries in simulated ABOiKT and in pre-clinical human models. Furthermore, re-expression of blood group antigens within 48 hours post enzymatic conversion did not elicit hyperacute rejection. However, significant research gaps exist in translating this novel technology from bench to bedside. The ECO-kidney holds great promise as a beacon of hope to vanquish the dreaded foe of ABO incompatibility, thus restoring equity and harmony in the realm of transplant.

May the ECO-kidney serve as a boon for every patient in the future, by transforming the tragedy of disease into the triumph of transplant!

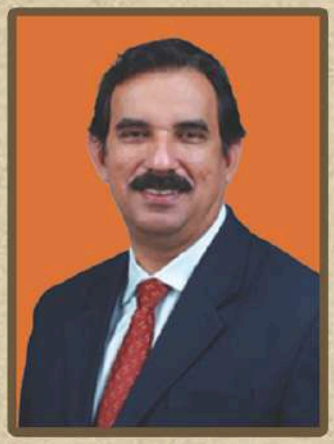
Welcome to ISNCON 2026

KOCHI

Dear Colleagues,

It is our great pleasure to invite you to **ISNCON 2026**, to be held in the beautiful coastal city of **Kochi, Kerala from 17 to 20 December 2026**. The annual meeting of the Indian Society of Nephrology has been organized on the banks of the serene backwaters of Lake Vembanad at the LuLu convention centre, Bolgatty, Kochi.

The meeting will showcase advances in clinical nephrology, kidney transplantation, dialysis, precision medicine, glomerular diseases, genetic kidney disorders, and emerging therapies that are shaping the future of kidney care. Delegates will be exposed to renowned national and international faculty, intense workshops covering all the latest advances in the field and get to experience the renowned Kerala hospitality in God's own country.



We warmly welcome you to join us in Kochi for ISNCON 2026

Save the date-
December 17-20 2026

We look forward to welcoming you to Kochi in 2026.

Dr George Kurian

Senior Professor, Dept. of Nephrology
Amrita Institute of Medical Sciences
Organising Secretary ISNCON 2026 Kochi

Crossword

1. MAGNESIUM: Cetuximab, an epidermal growth factor receptor (EGFR) inhibitor causes hypomagnesemia due to impaired stimulation of TRPM6 in the DCT.

2. CALVERT: The Calvert formula is used to calculate Carboplatin dose for a target AUC (carboplatin dose (mg) = target AUC (mg mL-1 min) × [GFR (mL/min) + 25 (mL/min)]). According to the ADDIKD guidelines, Directly measured GFR is preferred for dose estimation. If estimating GFR, BSA adjusted eGFR from the CKD-EPI equation is preferred.

3. ORCA-T: Orca-T is a precision cell therapy derived from stem and immune cells from allogeneic donors, where polyclonal regulatory T-cells are enriched and given at a targeted ratio with conventional T-cells, promoting immune tolerance and minimizing GVHD after alloHCT. It was found to have slower GFR decline compared to Methotrexate and Cyclophosphamide. It suggests that immune tolerant platforms may reduce post-alloHCT nephrotoxicity.

4. VINCRISTINE: Catharanthus roseus (Madagascar periwinkle) is the source for Vinca alkaloids, Vincristine and Vinblastine.

5. AXITINIB: The study showed that among patients with advanced renal-cell carcinoma, treatment with pembrolizumab plus axitinib resulted in significantly longer overall survival and progression-free survival than Sunitinib. Combination therapies of ICI and TKI such as Pembrolizumab-Axitinib, Pembrolizumab-Lenvatinib and Nivolumab-Cabozantinib and double ICI combinations such as Nivolumab-Ipilimumab are being increasingly used for better outcomes but raise the possibility of overlapping nephrotoxicities.

6. HYPERTENSION: Up to 80% of patients treated with VSPIs develop new or worsened hypertension and the AHA recommends that all patients undergo baseline screening for hypertension before the initiation of cancer treatment.

7. RCC (RENAL CELL CARCINOMA): Malignancy is reported to be the underlying etiology in ~7% of systemic AA amyloidosis cases. Renal cell carcinoma is the most common solid organ malignancy to cause AA amyloidosis; however, AA amyloidosis complicates only 2%–3% of renal cell carcinomas.

8. DOMAGK: Radiation nephropathy, first documented by Domagk in 1927, which develops over a long time after exposure to radiation, is characterized by proteinuria, azotemia, hypertension, and anemia. It causes cellular damage in all components of the kidney, including the glomerulus, blood vessels, tubular epithelium, and interstitials. Among them, the most striking morphological change was reported in glomerular endothelial cells, which detached from the basement membranes.

9. TOCILIZUMAB: T-cell engaging Bispecific Antibodies target a tumor associated antigen with one arm, and simultaneously bind to the CD3 subunit within the T-cell receptor (TCR) complex with the other arm and activate multiple immune pathways to eliminate the tumor cells. They are commonly associated with CRS. Treatment includes interrupting the infusion, glucocorticoids and in those with inadequate response, treatment with anti-IL6 MAb, Tocilizumab.

10. *IgA*: Elevations in SAG are usually due to a process leading to the accumulation of organic acids and subsequent decrease in bicarbonate without affecting chloride concentrations. The immunoglobulin can carry a charge that interferes with SAG in cancer patients with monoclonal gammopathy. For example, immunoglobulin A (IgA) is anionic and is positively correlated with SAG, and in one report an elevated SAG was discovered in 31% of IgA monoclonal gammopathies
11. IPILIMUMAB, a CTLA-4 inhibitor, was the first checkpoint inhibitor approved by the FDA (NDA approval date = March 2011). It was initially used in the treatment of metastatic melanoma.

12. SKIN: The skin is the earliest and most frequent target, and involvement begins with faint erythematous macules at any site, often the palms, soles, and pinna. Edema and tenderness often accompany. Pruritus is an uncommon complaint. As the acute GVHD evolves, the distribution of erythematous macules increases, becoming confluent especially over upper back. At a later stage, erythroderma and toxic epidermal necrolysis may ensue.

Quiz

Q1. (B) | **Q2.** (C) | **Q3.** (B) | **Q4.** (B)

Q5. (B) | **Q6.** (B) | **Q7.** (A) | **Q8.** (B)

Q9. (B) | **Q10.** (A) | **Q11.** (A) | **Q12.** (C)

Q13. (A) | **Q14.** (C) | **Q15.** (B)

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